

Editorial

Importance of Basic Biomedical Research in the Field of Cardiothoracic Surgery

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When scientists were sorting out the mysteries of cardiac and pulmonary cells, they may not have realized how their findings would apply to the use of surgical implants for coronary artery disease or to treatments for reducing high blood pressure or cardiac failure. Their primary target may focused to know how cells contract or how bio-molecules enter cells and change cellular activity. In this era filled with promise as well as debate, it is perhaps worth pondering why basic biomedical research is important and where we would be without it. In laboratories throughout most of the important medical centers, basic translational research activities are under way to better understand the pathogenesis of cardiovascular disease, to analyze how current treatments work, and to develop potential novel therapies. Sophisticated complex machines and surgical procedures stand ready to help the physician diagnose and treat patients-once they become sick, and often at higher expense. If we knew more, if we could prevent a disease entirely or cure it in its early stages, there would be tremendous savings of both money and misery.

For example, in United States, nearly 6 million individuals have been diagnosed with heart failure (HF). Of these, an estimated 550,000 patients are in advanced HF, defined as New York Heart Association (NYHA) Class IIIb/Class IV [1]. Left Ventricular assist device (LVAD) therapy has evolved into a standard therapy for patients with advanced HF, not only as a bridge to myocardial recovery or cardiac transplantation but also as a destination therapy [2,3]. The success of the LVAD therapy is attributed to the emergence of continuous-flow LVADs (CF-LVADs), which are small in size, low weight and very durable [4]. As studies continue to demonstrate the benefits and improving survival outcomes of CF-LVADs implantation worldwide, efforts to reduce the associated complications are needed [5]. Common complications after CF-LVAD implantation include infections, stroke, gastrointestinal bleeding and right HF [6]. As the horizon of support with these devices increases on the order of years, further basic science research and data will emerge on mitigation strategy on the long-term consequences of pump-blood component interaction and related clinical complications such as bleeding, infection and stroke.

Cardiothoracic surgeons still see the blood and gross anatomy of heart during operation progresses, but their minds are now focused on invisible structures. Platelets, leukocytes, complement, growth factors, interleukins, adhesion surface molecules, second messengers and transcription factors that may be disturbed by the pre-existing conditions, by the knife or by the biomedical device itself. Today's surgeons' now becoming implicit basic scientist and their laboratories are becoming as important as the operating theatre. However, due to the very busy schedule, the surgeons may not be able to cover all requirements for basic experimental work. Therefore, a close collaboration with basic science research scientist may fulfill the prospects of the clinician.

Studies on biomedical basic science research revealed that preexisting oxidative stress and high non-physiological mechanical shear stress generated by CF-LVADs may be responsible for platelet activation or abnormalities and has been regarded as the culprit of the increased thrombotic risk or bleeding in patients with mechanical circulatory support during HF condition [7-13]. Years ago, bioengineers use the relationship between shear stress/exposure time and hemolysis to guide the device design and to successfully mitigate the hemolysis problem in contemporary biomedical devices [14,15]. However, hemolysis alone is insufficient to elucidate the cause of thrombosis and bleeding associated with those devices. In addition, flow stasis and artificial surfaces also play an important role in device related thrombogenesis. These findings help the cardiothoracic surgeons and clinicians to introduce anti-thrombotic therapies targeting platelets and coagulation pathways to mitigate the thrombotic risk by inhibiting platelet activation, aggregation and adhesion, and/or coagulation enzymes. These therapies and the development of new anti-thrombotic medications have revolutionized the medical management of hypercoagulability, such as deep vein thrombosis, left atrial fibrillation, and significantly reduced thrombotic events in patients.

Now-a-days multiple basic science research groups throughout the nation targeting the role of non-physiological high shear stress generated by CF-LVADs which may enable mitigation of clinical complications, improve medical management and patient outcomes, and lead to the development of less traumatic blood contacting medical devices. In order to attack such major

diseases of today like heart disease, cancer, AIDS, arthritis, and diabetes, we need a broader base of knowledge. We need to know more about the specific cellular and molecular mechanisms involved in the process of these conditions. By providing this knowledge, basic biomedical researches form the foundation for advances in the diagnosis, treatment, and prevention of such diseases and also help cardiothoracic surgeons and clinicians to manage their patient.

REFERENCES

1. Miller LW. Left ventricular assist devices are underutilized. *Circulation*. 2011; 123: 1552-1558.
2. Birks EJ, Yacoub MH, Banner NR, Khaghani A. The role of bridge to transplantation: Should LVAD patients be transplanted?. *Curr Opin Cardiol*. 2004; 19: 148-153.
3. Birks EJ, Tansley PD, Hardy J, George RS, Bowles CT, Burke M, et al. Left ventricular assist device and drug therapy for the reversal of heart failure. *N Engl J Med*. 2006; 355: 1873-1884.
4. Xie A, Phan K, Yan TD. Durability of continuous-flow left ventricular assist devices: a systematic review. *Ann Cardiothorac Surg*. 2014; 3: 547-556.
5. Harvey L, Holley CT, John R. Gastrointestinal bleed after left ventricular assist device implantation: incidence, management, and prevention. *Ann Cardiothorac Surg*. 2014; 3: 475-479.
6. Wilson SR, Givertz MM, Stewart GC, Mudge GH. Ventricular assist devices the challenges of outpatient management. *J Am Coll Cardiol*. 2009; 54: 1647-1659.
7. Mondal NK, Sorensen EN, Hiiivala NJ, Feller ED, Pham SM, Griffith BP, et al. Intraplatelet reactive oxygen species, mitochondrial damage and platelet apoptosis augment non-surgical bleeding in heart failure patients supported by continuous-flow left ventricular assist device. *Platelets*. 2015; 26: 536-544.
8. Hu J, Mondal NK, Sorensen EN, Cai L, Fang HB, Griffith BP, et al. Platelet glycoprotein Iba α ectodomain shedding and non-surgical bleeding in heart failure patients supported by continuous-flow left ventricular assist devices. *J Heart Lung Transplant*. 2014; 33: 71-79.
9. Mondal NK, Sorensen E, Hiiivala N, Feller E, Griffith B, Wu ZJ. Oxidative stress, DNA damage and repair in heart failure patients after implantation of continuous flow left ventricular assist devices. *Int J Med Sci*. 2013; 10: 883-893.
10. Chen Z, Mondal NK, Ding J, Koenig SC, Slaughter MS, Wu ZJ. Paradoxical Effect of Nonphysiological Shear Stress on Platelets and von Willebrand Factor. *Artif Organs*. 2016; 40: 659-668.
11. Chen Z, Mondal NK, Ding J, Koenig SC, Slaughter MS, Griffith BP, et al. Activation and shedding of platelet glycoprotein IIb/IIIa under non-physiological shear stress. *Mol Cell Biochem*. 2015; 409: 93-101.
12. Ding J, Chen Z, Niu S, Zhang J, Mondal NK, Griffith BP, et al. Quantification of Shear-Induced Platelet Activation: High Shear Stresses for Short Exposure Time. *Artif Organs*. 2015; 39: 576-583.
13. Chen Z, Mondal NK, Ding J, Gao J, Griffith BP, Wu ZJ. Shear-induced platelet receptor shedding by non-physiological high shear stress with short exposure time: glycoprotein Iba α and glycoprotein VI. *Thromb Res*. 2015; 135: 692-698.
14. Bernstein EF, Blackshear PL Jr, Keller KH. Factors influencing erythrocyte destruction in artificial organs. *Am J Surg*. 1967; 114: 126-138.
15. Indeglia RA, Shea MA, Varco RL, Bernstein EF. Mechanical and biologic considerations in erythrocyte damage. *Surgery*. 1967; 62: 47-55.

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